

Branch Retinal Artery Occlusion Associated with Toxoplasma Retinitis

Dear Editor,

Branch retinal artery occlusion (BRAO) usually manifests as an embolic obstruction at vessel bifurcations in older individuals, most commonly originating from the carotid arteries as a result of atherosclerotic disease. BRAO rarely occurs in patients younger than 40 years of age and is more commonly a result of an underlying hypercoagulable state, autoimmune disease, or active infection. Toxoplasma infection in the eye manifests as a necrotizing chorioretinitis that can cause a variety of retinal vascular changes [1]. However, reports of retinal arterial occlusions caused by toxoplasma retinitis are rare in the literature. Herein, we report a case of BRAO in a healthy younger man associated with active toxoplasma chorioretinitis.

A 38-year-old man developed a complete loss of the infe-

six weeks after the onset of his symptoms. The patient was otherwise healthy with no significant past medical or ocular history. His best-corrected visual acuity was 20 / 40 in the right eye and 20 / 20-1 in the left eye with normal intraocular pressures in both eyes. Examination of the left eye was normal. Anterior segment examination of the right eye showed no anterior chamber inflammation. There were 1+ anterior vitreous cells. Fundoscopic examination showed a pigmented chorioretinal scar superonasal from the fovea with adjacent peripapillary whitish retinal infiltrate and segmental retinal periarteritis (Kyrieleis plaques) involving superonasal and superotemporal retinal arterioles (Fig. 1A). Humphrey visual fields Humphrey visual fields 24-2 (Carl Zeiss Meditec, Dublin, CA, USA) showed an inferior altitudinal defect in the right eye with enlargement of the blind spot; HVF OS was normal. Optical coherence tomography through the active retinitis lesion showed full-thickness retinal hyperreflectivity and diffuse inner retinal thinning in the superior macula consistent with arterial occlusion (Fig. 1B). Optical coherence tomography angiography demonstrated superficial and deep capillary plexus nonperfusion in the territory of the supero-

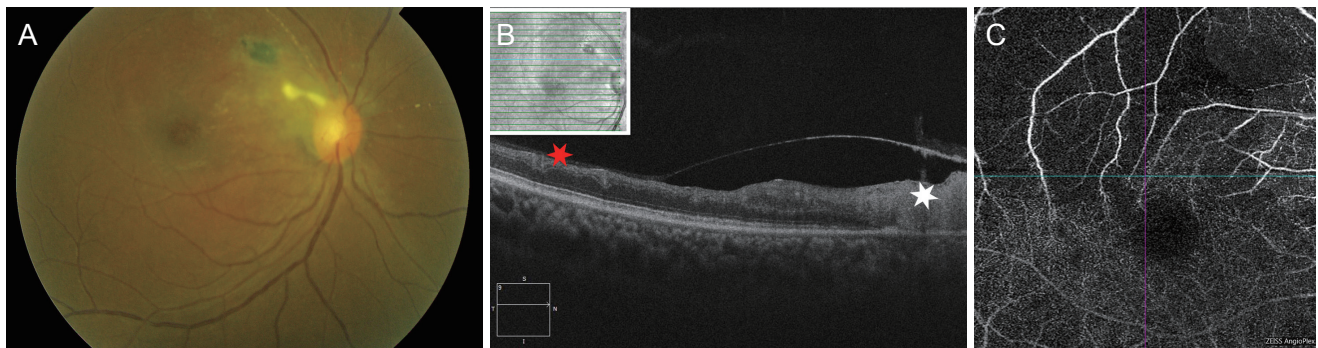


Fig. 1. Clinical findings. (A) Color fundus photo. Pigmented chorioretinal scar located superonasal from the fovea with adjacent peripapillary retinal infiltrate. Segmental retinal periarteritis involving superonasal and superotemporal arterioles is apparent. (B) Optical coherence tomography of the infiltrative lesion shows full-thickness retinal hyperreflectivity (white star) and diffuse inner retinal thinning in the superior macula consistent with arterial occlusion (red star). (C) Optical coherence tomography angiography of the deep capillary plexus of the right eye showing nonperfusion.

rior field of vision in the right eye without any spontaneous improvements. He presented to our clinic for evaluation

temporal retinal artery (Fig. 1C).

Laboratory workup was significant for positive immunoglobulin G titer of 238.0 IU/mL (reference <4.0 mL non-detection) to *Toxoplasma gondii*, indicating previous infection. Immunoglobulin M levels were normal (<1 : 10).

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Syphilis antibody test and T-SPOT.TB assay (Oxford Immunotec, Abingdon, UK) test were negative. The patient was placed on a 6-week course of trimethoprim/sulfamethoxazole (1,600/320 mg orally twice daily) and active chorioretinitis resolved on the subsequent visit. However, inferior altitudinal visual field corresponding to arterial occlusion persisted.

Toxoplasma infection in the eye manifests as a necrotizing chorioretinitis often adjacent to old pigmented chorioretinal scar, vitritis, and anterior chamber inflammation. Clinical findings of active retinitis adjacent to a pigmented chorioretinal scar and segmental retinal periarthritis are highly suggestive of ocular toxoplasmosis, and diagnosis can be confidently made based solely on fundusoscopic examination. Nevertheless, serologic studies help confirm the diagnosis and rule out masquerading infectious agents such as tuberculosis or syphilis, which may also present with active retinitis and retinal periarthritis.

Ocular toxoplasmosis can cause a variety of retinal vascular changes, including segmental retinal periarthritis [1], which is a non-specific finding associated with active or recently healed toxoplasma chorioretinitis [2]. Its pathogenesis is not fully elucidated, however, recent multimodal imaging demonstrated inflammatory deposits within the vessel wall sparing the lumen, suggestive of endothelial inflammation [3]. As the lumen of the vessel is spared, angiographic studies of the segmental retinal periarthritis have confirmed normal vascular perfusion and showed no evidence of vascular obstruction [3,4]. Thus, segmental retinal periarthritis is not a source of retinal arterial occlusion. Retinal vascular occlusion associated with ocular toxoplasmosis is thought to be caused by active necrotizing retinitis overlying or adjacent to retinal vessels resulting in vasoconstriction and increased blood viscosity [2] that may lead to thrombosis of the involved vessel. BRAO-associated with toxoplasma retinitis is rarely described in the literature [2,5], but previous reports share in common resolution of infectious foci with prompt antibiotic coverage with subsequent steroid treatment. However, there is a conflict-

ing evidence in the literature if antibiotic therapy results in better visual outcomes in patients with toxoplasma retinitis.

This case highlights the local destructive nature of the acute chorioretinitis leading to retinal artery occlusion. Retinal arterial occlusions may rarely complicate toxoplasma retinitis and result in significant visual acuity or visual field loss despite the resolution of active retinitis.

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Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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